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Guidelines for Surface Water Quality

Vol. 1 Inorganic Chemical Substances

MERCURY

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Abstract

A literature survey was carried out on the toxic effects of mercury and mercury compounds on human health, aquatic life, plants and livestock. The information is summarized in this publication. From it, maximum mercury concentrations in water at which toxic effects will not appear are recommended.

Résumé

On a effectué une étude de la documentation portant sur les effets toxiques du mercure et de composés du mercure sur les humains, les organismes aquatiques, les plantes et le bétail. Ces données sont résumées dans cette publication. En se basant sur ces données, on propose des valeurs des concentrations maximales de mercure dans l'eau auxquelles on n'observe pas d'effets toxiques.

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RECOMMENDED WATER QUALITY OBJECTIVES FOR MERCURY

Uses	Recommended objectives
Raw public water supply	0.001 mg/L as total mercury*
Aquatic life and wildlife	
Aquatic life	0.0001 mg/L as total mercury* to protect consumers of fish 0.0002 mg/L as total mercury* where fish are not eaten, to protect aquatic life
Wildlife	0.003 mg/L as total mercury* (see section on mercury content of fish for fish eaters)
Agricultural water supply	
Livestock watering	0.003 mg/L as total mercury*
Irrigation water	No limit recommended
Recreation and aesthetics	
Recreational water	0.001 mg/L as total mercury* (see section on aquatic life for mercury content of fish)
Aesthetics	No limit recommended
Industrial water supplies	
Food and drink industries	0.001 mg/L as total mercury*

*Total mercury is determined in an unfiltered sample and includes all forms of mercury, inorganic as well as organic.

Guidelines for Surface Water Quality
Vol. 1. Inorganic Chemical Substances. Mercury

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SOURCES AND USES

Mercury in metallic (elemental) form or as inorganic or organic compounds is present throughout the whole ecosystem. Figure 1 represents schematically mercury distribution and the transfer of mercury which takes place between the various parts of the ecosystem. The diagram and the values presented have been adapted from Hugunin and Bradley (1975a), Jonasson and Boyle (1972), and Wollast *et al.* (1975). In addition to the processes represented in Figure 1, there are many other transfers occurring within each part of the ecosystem.

World mine production, which was estimated at 9000 tonnes in 1975, came from mercury ores with cinnabar (HgS) as the primary mercury mineral (George 1976). The only Canadian producer, the Pinchi Lake mine of Cominco Ltd. in British Columbia, had an output of approximately 414 tonnes in 1975. In addition to the Pinchi Lake mine output, Canada imported 74 tonnes that year (George 1976).

World consumption of mercury in 1975 was approximately 8620 tonnes, the same as the 1974 consumption. This lack of growth and in some countries, e.g., the United States and Canada, decline in mercury consumption, were explained as partly due to environmental concerns (George 1976). The Canadian consumption in 1974 was 38 tonnes, a sharp decline from a peak of 154 tonnes in 1970.

Uses of Mercury

The main uses of mercury are in the electrical industry (batteries, high-intensity discharge lamps, fluorescent lamps, rectifiers, oscillators, switches); the chemical industry (electrolytic preparation of chlorine and caustic soda, paints, fungicides, catalysts); industrial and control instruments; general laboratory uses; and medicine (dentistry and pharmaceutical products) (Hugunin and Bradley 1975a).

In Canada, of the 38 tonnes consumed in 1974, 72% went to the chemical industry, 15% to electrical apparatus and the remaining to other uses (George 1976).

Losses to the Environment

The main pathways by which the mercury reaches the environment and the estimated amounts are given in Table 1.

The losses of mercury in Canada from the burning of fuels and from other uses were estimated at 51 tonnes in 1970, when the consumption was at its peak (154 tonnes) (Environment Canada 1973, p. (iii)).

Table 1 shows that by far the largest amount of mercury released in the environment on a global basis, approximately 80%, comes from uncontrollable natural sources. In given local conditions anthropogenic sources might be more important than natural sources.

Table 1. Pathways by which Mercury Enters the Ecosystem (Data from Lagerwerff 1972, p. 609)

Pathway	Estimated amount of global emissions (tonnes/yr)
Physical and chemical breakdown of rocks and minerals containing Hg	230*
Natural evaporation	36 000
Burning of fossil fuels (coal, petroleum)	5 000
Losses related to uses of mercury	3 000†
Total	44 230

*Other values found in the literature are 800 tonnes/yr (Gavis and Ferguson 1972) and 3000 tonnes/yr (Hugunin and Bradley 1975a).
†Estimated at 25% to 50% of the world production. The middle range was used here.

MERCURY IN THE AQUATIC ENVIRONMENT

General

The distribution of mercury in the aquatic ecosystem, which comprises the hydrosphere and parts of the biosphere and lithosphere, is given in Table 2.

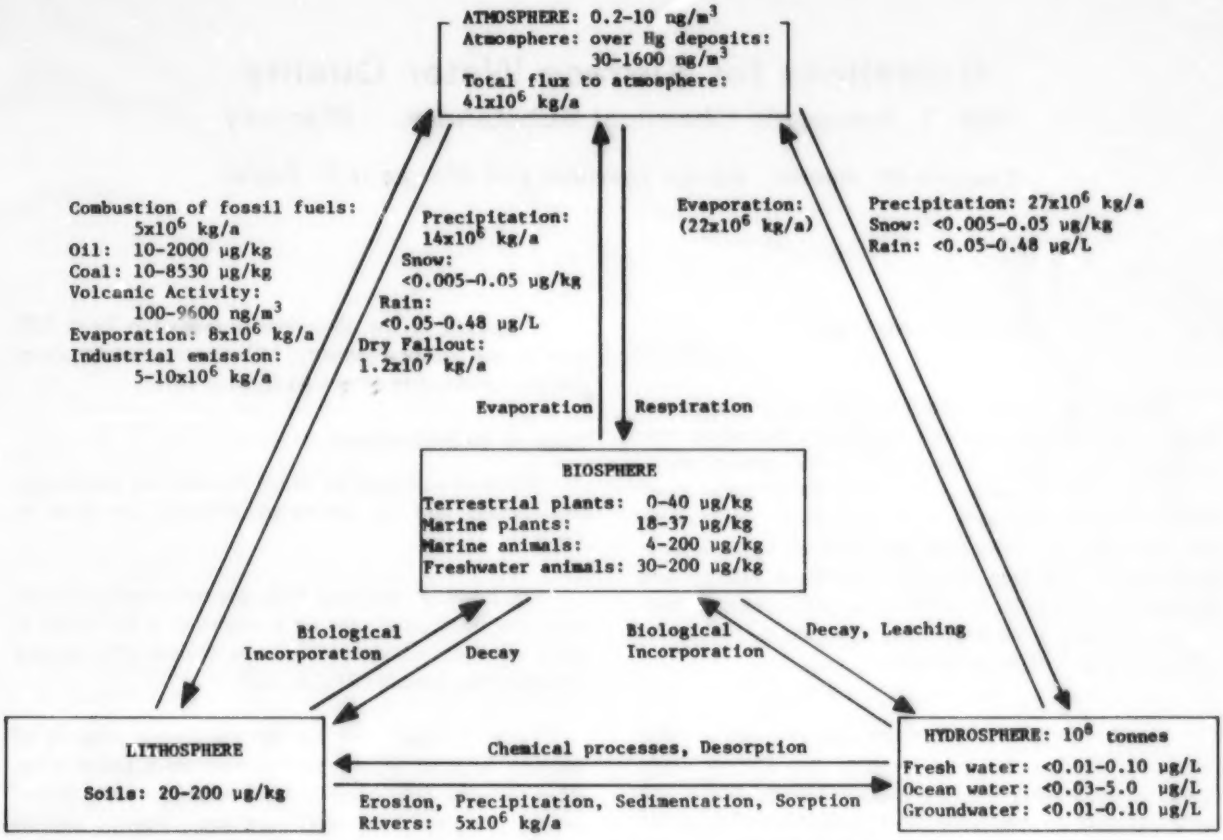

Figure 1. The distribution and the natural processes for transfer of mercury between parts of the global ecosystem (adapted from Hugunin and Bradley 1975a; Jonasson and Boyle 1972; Wollast *et al.* 1975).

In a study of the Ottawa River (Kudo *et al.* 1977), the distribution of mercury was found to be 97.2% in bed sediment, 2.5% in water, 0.3% in suspended solids, and 0.02% in biota. This is in good agreement with the Swedish data in Table 2.

Table 2. Distribution of Mercury in the Aquatic Ecosystem (Data from Jernelöv and Lann 1973)

Compartment	Total mercury (%)	Methylmercury (%)
Sediment	90-99	1-10
Water	1-10	<1
Biota	<1	90-99

The transfer of mercury in the aquatic system can be separated into geographical movement and compartmental transfer. The transfer between compartments is independent of geographical movement. The geographical movement is carried out exclusively by nonbiological mechanisms (Miller and LaHam 1976).

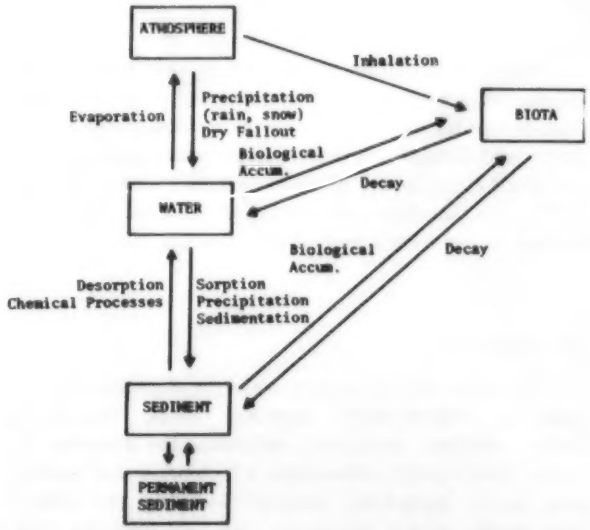

Figure 2. The transfer processes of mercury in an aquatic environment.

Table 3 shows the percentage of geographical mercury transport by the various compartments. Water is by far the most important vector in this process, although at any one time it contains a very small percentage of the total mercury. Figure 2 represents schematically the transfer processes taking place in an aquatic environment.

Table 3. Geographical Movement of Mercury in an Aquatic Environment (Calculated from Miller and Latham 1976, p. 1.11)

Compartment	Percentage of total mercury transported
Water	87.3
Suspended particles	9.5
Sediment	3.2

Mercury in Water

Mercury is present in water in many different forms. Attempts to calculate the amounts of different mercury species in water were not successful on account of the complexity of the natural waters and the low concentration of mercury (Shin and Krenkel 1976).

Inorganic mercury in natural waters was shown to be rapidly and efficiently transferred into the sediment, almost 100% in 5 minutes, and was little affected by variation in pH (Ramamoorthy and Rust 1976). The uptake rates of inorganic mercury by a variety of Ottawa River sediments ranged mostly between 5 and 10 cm/day (in other words, 1 cm^3 of bed sediments would retain each day the amount of mercury present in an overlying column of water 5 to 10 cm high) (Kudo and Hart 1974). No significant difference in the uptake rates was observed between aerobic and anaerobic conditions. The uptake phenomena were surface dependent, and the depth of the bed sediment affected was less than 0.1 cm. The opposite transfer, sediment to water, was volume dependent (Kudo *et al.* 1975). The uptake rate of methylmercury ions was higher than the value for inorganic mercury by about 10% (Mortimer and Kudo 1975).

Mercury present in the aquatic ecosystem takes part in a multitude of reactions. Some general schemes have been proposed; one of them has been reproduced in Figure 3. Temperature, the availability of mercury ions, the metabolic activity of methylating organisms, and the presence of strong organic chelators affect the chemical reactions of mercury in water and sediments (Karbe *et al.* 1975).

Probably the two most important reactions of mercury in water are (i) the exchange of mercuric ions for other cations in insoluble sulphidic minerals (because mercuric sulphide has the lowest solubility of all sulphides) and (ii) the reaction of mercuric ions with bioproduced hydrogen sulphide. Both of these reactions occur mainly in sediments and lead to the formation of insoluble mercuric sulphide (HgS). Similarly, the reaction of the methylmercury ion $(CH_3Hg)^+$ with the sulphide ion (S^{2-}) leads to the formation of $(CH_3Hg)_2S$, the second most insoluble sulphide (Clarkson 1972).

The formation of insoluble sulphides is one of the detoxication processes taking place in natural waters. A second detoxication process of the aquatic environment is the evaporation of mercury compounds from the water surface (Johnson and Braman 1974). Note that this second process contributes to the airborne transport of mercury.

Ecologically important methylmercury is formed from the mercuric ion by enzymatic and other reactions under aerobic or anaerobic conditions (Hugunin and Bradley 1975a; Bisogni and Lawrence 1975; Rissanen 1974). Only 0.1% per year of mercury is converted to methylmercury (Jernelöv *et al.* 1975).

The natural levels of mercury in Canadian waters vary from area to area and are usually close to the detection limit, but streams and rivers near mercury deposits may contain up to 0.1 mg/L. The current detection limit for the analytical method usually employed to measure mercury in water is 0.05 µg/L mercury.

Mercury in Sediments

Mercury accumulates in sediments from natural sources and from man's activities.

The content of mercury in sediments is highly variable. The lowest values, in the region of 0.001mg/kg, are found in the sand fraction, and the highest mercury content is generally found in sediments rich in organic material.

Mercury is present in sediments in different forms. It occurs (i) as particles of mercuric sulphide (either from erosion of minerals containing mercuric sulphide or precipitated from the mercuric sulphide ion), (ii) as droplets of metallic mercury (from natural sources or as a result of reduction of mercury compounds) or (iii) chemisorbed or adsorbed on either organic or inorganic materials as mercuric ion and methylmercuric ion (Krenkel 1974).

The release of mercury from sediments is slow (Jacobs and Keeney 1974). The desorption rate of mercury from Ottawa River sediments ranged from 0.1 to 1.0 $ng/cm^2 \cdot day$ and was volume dependent (Kudo *et al.* 1975).

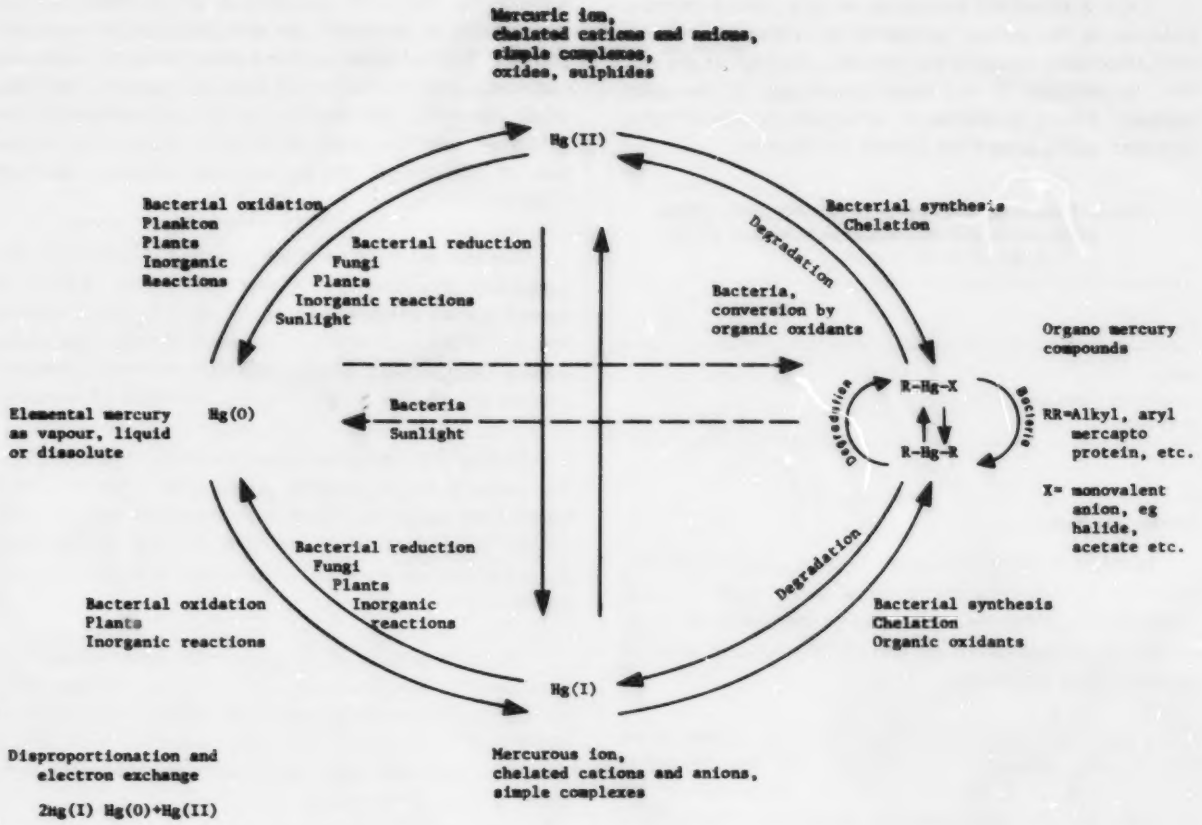

Figure 3. Cycle of mercury interconversions in nature (from Jonasson and Boyle 1972).

RATIONALE

Raw Public Water Supplies

Mercury in Drinking Water

The Department of National Health and Welfare has recommended a limit of 0.001 mg/L of total mercury in drinking water in Canada (NHW 1977). The following considerations and assumptions led to the establishment of this limit.

(i) The first symptoms of mercury poisoning appear in a small percentage of the population at an estimated long-term total daily ingestion of 0.004 mg of methylmercury per kilogram of body weight. For a person weighing 70 kg this means an ingestion of 0.28 mg/day.
(ii) A safety factor of 10 was used.
(iii) The average total daily intake of mercury by an adult Canadian, based on food consumption patterns and on the average mercury content of inhaled air and drinking water, is 0.016 mg.
(iv) An additional safety factor was introduced by assuming that the mercury is present only as methylmercury, its most dangerous form.

Thus, for a person of an average weight of 70 kg, the tolerable total daily intake would be 0.028 mg. By permitting a maximum limit of 0.001 mg/L in drinking water, the daily ingestion owing to water would be around 0.002 mg or approximately 10% of the tolerable intake level.

Mercury in Raw Public Water Supply

In establishing a limit for mercury concentration in the raw supply for drinking water, the elements that must be considered are (i) the maximum permissible concentration of mercury in drinking water and (ii) the removal of mercury by water treatment processes.

Conventional water treatment processes are not able to remove the very low concentrations of methylmercury (Water Research Centre 1976, p. 28). Thus, the maximum limit for total mercury concentration in raw supplies for

drinking water must be the same as that in the drinking water, 0.001 mg/L.

Aquatic Life and Wildlife

Levels in Organisms

A number of studies have determined the form, concentration factor and the location of mercury in the bodies of aquatic organisms.

The form of mercury most commonly found in fish is methylmercury. In Canadian inland waters uncontaminated by loss of mercury from industrial sources, fish usually have total mercury levels below 0.5 mg/kg (wet weight basis), the safety limit set for human consumption (Lockhart *et al.* 1972; Armstrong and Hamilton 1973; Environment Canada 1976).

Experimental exposure of brook trout to 0.03 µg/L of methylmercury in water resulted in the accumulation of 0.7 mg/kg wet weight total body burden (1.0 mg/kg wet weight in muscle) after 39 weeks (McKim *et al.* 1976). Other authors have quoted figures giving highly varied magnification factors for mercury accumulation in aquatic organisms. Kudo (1976) found a 600% difference between fish of the same species under natural conditions, thus indicating that caution must be exercised when studying fish caught in natural waters to ensure that a representative sample is obtained. Eighty to 93% of the total mercury in fish muscle as well as 50% of the total mercury in benthic macroinvertebrates was methylmercury (Hildebrand *et al.* 1976; Scherer *et al.* 1975).

The rate of uptake of mercury depends on the type of exposure, whether experimental or natural (Järvenpää *et al.* 1970; McKim *et al.* 1976), and this should be taken into account when considering experimentally derived accumulation factors.

The route of entry of mercury varied according to the species of fish and its mode of living, but the mechanisms for entry into the body have not yet been fully explained. Mercury can enter via the alimentary canal, the gills, and through the skin. It is thought that proportionately more mercury enters via the gills when oxygen is low due to an increased ventilation rate (Lloyd 1961).

Jernelöv and Lann (1971) studied the uptake of mercury in fish in three Swedish lakes. Mercury accumulation through a food web tended to cause a basic mercury concentration in the predators which was of the same magnitude as that of their prey. More mercury was added to this basic level, as it also entered the predator fish directly from the water. The percentage accumulation from food was constant for the predatory fish (80%) in the three lakes, but direct accumulation from the water was more important (75%) for bottom feeders. McKim (1974) reported concentration factors in fish in excess of 10 000 times the amount of mercury in the surrounding water.

The accumulation of mercury by organisms was said to be unrelated to their trophic level and to depend on other factors such as metabolic rates (Hannerz 1968). Contrary to this, Holm and Cox (1974) indicated that macroinvertebrates took up mercury to varying degrees depending on their trophic level and whether they fed on the bottom or in the water column. Bottom feeders (of detritus or benthos) had ten times as much mercury as planktivores (Hamilton 1972).

The uptake of mercury ($^{203}HgCl_2$) by aquatic macrophytes was related to the concentration of mercury in the water. Mercury was adsorbed onto the plant surface and absorbed into the tissues. The submerged parts generally contained more mercury than the emersed parts. There was very little uptake of mercury from the sediments by roots (Eriksson and Mortimer 1975).

The accumulation of mercury in aquatic life and humans was also discussed in a number of studies (Wojtalik 1971; National Water Quality Laboratory, Duluth 1974; Schroeder 1974; Albert *et al.* 1973; and *Nord Hyg. Tidskr.* 1971).

Aquatic Life – Toxicity

There have been many studies on the toxic effects of mercury on aquatic organisms, but the majority of researchers used concentrations which had an obvious effect on the organism in question.

The growth of *Chlamydomonas* sp. (Chlorophyta) was affected by methylmercury, and severe growth retardation occurred at 0.02 µg/L (Lock 1975).

McKim *et al.* (1976) studied the long-term effects of methylmercuric chloride on three generations of brook trout (*Salvelinus fontinalis*). No significant effects on the number of viable eggs, survival, growth or reproduction by the second generation trout were noted for 0.03, 0.09, and 0.29 µg/L. A possible effect was observed at 0.93 µg/L of methylmercury, which was shown by a low ratio of the mean estimated weight of young per test female compared with the controls. There were also effects on spawning fish at this level. The young third generation trout were not affected by the three lowest concentrations of methylmercury in the 12-week period after hatching, although the ratios of the mean estimated weights of 12 week old young per female were less than the control at all concentrations tested. The most sensitive stages of the trout to methyl-

mercury were the early life history stages, embryo-alevin-juvenile, rather than the older juvenile. The maximum acceptable toxicant concentration (MATC) occurred between 0.93 µg/L, where there was some effect, and 0.29 µg/L, the level of no effect at pH 7.5 and water hardness of 45.0 mg/L (as $CaCO_3$). An average 96-h LC_{50} of 75.0 µg Hg/L was obtained in continuous flow bioassays with yearling and juvenile trout. This figure was used to determine the application factor (AF = MATC/96-h LC_{50}), which was between 0.013 and 0.004. The lower application factor could be applied to 96-h LC_{50} results for other species of fish if sublethal data were not available. It must be kept in mind that some fish may be more sensitive than the species (brook trout) used to derive the AF and that AF's do not take the existing water quality into account. As behavioural, physiological and toxicological effects become better known application factors and acute mortality tests will become less significant.

The sensitivity of early life history stages to metals has also been shown in other species (McKim and Benoit 1971; 1974; Pickering and Gast 1972; Eaton 1973).

As mercury can be present in a number of forms, due to both chemical and microbial actions, one form cannot be used alone for recommending a limit. Therefore total mercury is used.

Since methylmercury is quite a small percentage of total mercury in water (IJC 1976), the level of total mercury of 0.2 µg/L proposed as the objective for the Great Lakes would contain only a fraction of the level of methylmercury which has been shown to have a significant effect on a number of organisms: 0.07 µg/L for fathead minnows (Mount 1974), between 0.29 and 0.93 µg/L for brook trout (McKim *et al.* 1976), and 0.02 µg/L for *Chlamydomonas* (Lock 1975). One major concern is the accumulation of mercury in fish to levels that are known to affect humans adversely.

Olson *et al.* (1975), in studying the uptake of mercury by fathead minnows (*Pimephales promelas*), showed that fish in the lowest test concentration of 0.018 µg/L methylmercury accumulated a total body concentration of 1.47 mg/kg. Levels of 0.03 µg/L methylmercury in water produced mercury residues in edible parts of fish above the 0.5 mg/kg limit for human consumption, an accumulation factor of at least 17 000 (McKim *et al.* 1976). Experiments with other fish species led to the same conclusion, i.e., very low, nontoxic levels of mercury in water can cause significant bioconcentration.

Since the muscle concentrations mentioned above did not produce obvious detrimental effects on the fish, the level of mercury in fish set for human consumption (0.5 mg/kg Hg wet weight) would be reached before the fish were significantly affected. Yet the work of two Swedish researchers has indicated an effect on the central nervous system of fish with low levels of mercury in the muscle. A negative correlation between the amount of methylmercury in the muscle and the ability of the fish to compensate for torque when swimming in rotatory water was reported by Lindahl and Schwanbom (1971a, b). The effect of methylmercury on the fish was significant at 0.005 mg/L methylmercury in water and at corresponding levels (0.232 mg/kg) of mercury in muscle. Higher methylmercury levels in muscle and water were also studied. This is evidence of a possible decrease in the fitness of fish to compete successfully when contaminated with low levels of mercury.

In order to prevent the accumulation of mercury in fish to levels dangerous to consumers of fish, it would appear that the level in water should be no higher than 0.01 µg/L methylmercury, the most toxic species of the metal. Assuming that methylmercury is not more than 10% of the total mercury (Table 2), this figure leads to a level of 0.1 µg/L total mercury.

Effects of Other Components of the Aquatic Environment on the Toxicity of Mercury

Bioassays provide essential information for evaluating the significance of hazardous chemicals in the environment, but extrapolation from laboratory information to the environment depends on knowledge of the natural environmental conditions.

MacLeod and Pessah (1973) studied the effect of different temperatures (5°C, 10°C, 20°C) on the toxicity and rate of accumulation of various forms of mercury in fish (*Salmo gairdneri*). An increase in temperature decreased the time to death, and a greater biomagnification occurred at 20°C than at 10°C at the same concentration of mercury. This may indicate that the higher temperature potentiates the depressant action of mercury. A 10°C rise in temperature increased the toxicity of the mercuric ion to carp-like fish threefold (Rehwoldt *et al.* 1972). Low levels of dissolved oxygen increased the toxicity of heavy metals due to an increase in the rate of respiratory flow (Lloyd 1961).

Mercury levels in fish in Swedish lakes were generally higher in oligotrophic lakes than in eutrophic lakes with the same degree of contamination. This may be due to the larger biomass in a eutrophic lake producing dilution of the mercury pool throughout the biota. There is usually a greater efficiency in the transfer of organic materials between trophic levels in an oligotrophic lake; this may also explain the higher levels of mercury in fish from such lakes. It was

suggested that as more decomposition occurred in eutrophic lakes more of the mercury in lower trophic levels would be lost through death and decomposition and not in organisms caught as prey, thus passing to the higher trophic levels (Jernelöv 1972). Larsson (1971) found a negative correlation between the pH of the water and mercury levels in fish.

Mixtures of heavy metals have been shown to affect the toxicity of each other. There is antagonism between mercury and copper. In mixtures where the proportions were 80% methylmercury to 20% copper and 40% methylmercury to 60% copper, the copper seemed to protect against the toxicity of methylmercury. This is similar to the protective action of zinc against mercury in mammalian embryos (Roales and Perlmutter 1974). Some initial investigations into the effect of methylmercury and copper on immune response in fish showed that the two toxicants alone or together resulted in an apparent decrease in the immune response to both the infectious pancreatic necrosis (IPN) virus and *Proteus vulgaris* (Roales 1973). These topics have only been touched and further investigation is needed.

Northern creek chubs (*Semotilus atromaculatus*) pretreated with selenium (3 mg/L for 48 h) had lower mortality rates when subsequently exposed to mercury (as mercuric chloride) than fish without pretreatment. Selenium protection did not appear to be due to a decrease in the amount of mercury in the body up to exposure levels of 0.07 mg/L Hg. At higher mercury levels, 0.1, 0.13 and 0.16 mg/L, the mercury body burdens were less in selenium-pretreated fish than in the controls. The amount of mercury accumulated in the fish correlated with the amount of mercury in water and gave a concentration factor of 32 over 48 h (Kim *et al.* 1977). This study, done under laboratory conditions, suggested an antagonistic effect of selenium on mercury toxicity. A study by Huckabee and Griffith (1974), using the percentage hatch of carp eggs, indicated a synergistic action between mercury and selenium. It was suggested that the percentage of eggs that hatched when exposed to concentrations of mercury and selenium combined was lower than the percentage that hatched when exposed to the same concentrations of either mercury or selenium alone. As both selenium and mercury have an affinity for SH groups, they may have reacted directly with the SH groups in the outer membrane of the egg to kill it. In contrast with the work by Huckabee and Griffith, most studies with multicellular organisms (not eggs) have indicated that mercury and selenium are less toxic when they are together and their interaction needs further study. Even if free-swimming fish are not adversely affected by low levels of selenium and mercury existing in the environment together, the reproductive cycle may be disrupted by the eggs not being able to hatch.

Two possible mechanisms for selenium protection against mercury toxicity were proposed by Ohi *et al.* (1976). The authors quoted work by Ganther *et al.* (1972) that selenium may increase non-toxic binding sites which have a great affinity for heavy metals and hence divert them from their usual targets and work by Stillings *et al.* (1974) that selenium facilitates the conversion of methylmercury to a less toxic form. It was suggested that the second mechanism occurred in nature, as most of the mercury in animals at the top of the food web in the ocean was not in the form of methylmercury. Selenium was also present in large amounts in these animals (Koeman *et al.* 1973; Hagiwara 1974; Tamura *et al.* 1975).

Hard water had an antagonistic effect on the toxicity of mercury chloride to the protozoan *Tetrahymena pyriformis* (Carter and Cameron 1973), but Tabata (1969) did not find any significant effects of hardness on the toxicity of Hg^{2+} to *Daphnia*, rainbow trout, carp or Japanese killifish.

Some phosphatase enzymes involved in cellular transport processes require K^+, Na^+ and Mg^{2+} or Mg^{2+} alone for their activity (White *et al.* 1973). It was suggested that mercury uptake into cells was linked to a metabolic process requiring Mg^{2+}, as the presence of magnesium ions seemed to enhance the uptake. Inhibition of mercury uptake by respiratory inhibitors such as CN^- also suggested that metabolism was involved (Fujita *et al.* 1976).

Recommended Objective for Aquatic Life

The concentration of total mercury in water to protect aquatic life should not exceed 0.0002 mg/L. To prevent accumulation of mercury in fish to levels beyond those recommended for consumption by humans (0.5 mg/kg) and by fish-eating wildlife (see next section), however, a level of 0.0001 mg/L total mercury in water would have to be the recommended limit so that fish living in those waters could be eaten.

In waters where fish play an important role in the economy of the area, the lower level of 0.0001 mg/L should be considered. If fish from the waters in question are not eaten, 0.0002 mg/L total mercury could be considered.

Wildlife – Toxicity

The safe level of methylmercury in the diet of birds is close to 0.5 mg/kg. Consequently, fish-eating birds should be protected if the total mercury in fish does not exceed 0.5 mg/kg (IJC 1976). Fimreite (1974) found that the level of mercury in the liver of surface-feeding birds, which consume mostly vegetation and macroinvertebrates, was considerably less (<10 mg/kg total Hg) than in the liver of

fish eaters (100 mg/kg total Hg). Mallard ducks in the mercury-contaminated Lake St. Clair area had an estimated consumption of 0.008 mg total Hg/day, while ducks with a predominance of fish in the diet consumed 0.175 to 0.681 mg total Hg/day depending on the season (Ford and Prince 1975). The higher concentrations could be toxic if consumed over a long period. The amount of mercury in the egg is thought to be related to mercury in the food rather than to the amount stored in the tissues (Gilbertson 1974). The percentage hatch was lower in eggs with mercury levels above 0.5 mg/kg Hg (Fimreite *et al.* 1970). Mercury did not appear to affect eggshell thickness (Fimreite *et al.* 1970; Peakall and Lincer 1972; Heinz 1974).

Elevated levels of mercury have been reported in the hair of otter and mink in Georgia (Cumbie 1975), and in Sweden large declines in numbers have been reported for the last decade in populations of otter (Borg 1969, 1975; Borg *et al.* 1971). One case of clinical illness in a wild mink, which normally has quite a high percentage of fish in its diet, was reported by Wobeser (1976). The levels of mercury in its organs and fur were higher than those measured in experimentally poisoned mink. This is thought to be the first recorded case in North America.

A strong positive correlation has been found between the concentrations of selenium and mercury in tissues of various vertebrates including man. It has been suggested that the co-accumulation of selenium provides some sort of protection against mercury, but the mechanism is not known (Bryan 1976). Recent experiments have shown that selenium salts acted directly with both dimethylmercury and methyl-B_{12} to give dimethylselenide as the major product. Exhalation of the volatile selenium compound was regarded as a detoxification mechanism, and it seems that this is one way of removing the methyl-group and decreasing the toxic effects due to methylmercury (Wood *et al.* 1975). The combination of vitamin E and selenium was more effective than selenium alone in protecting against the effects of mercury in Japanese quail (Welsh and Soares 1976) (See section on livestock).

Recommended Objective for Wildlife

To protect fish-consuming animals (and man), the total mercury in fish should not exceed 0.5 mg/kg on a wet weight basis. This recommended level of 0.5 mg/kg in fish would accumulate from a very low level of mercury in water, since magnification factors have been shown to be as high or higher than 10 000 (EPA 1976, p. 185). Thus 0.5 mg/kg mercury could come from water containing as little as 0.05 µg/L mercury, much below the level of mercury that is recommended as the safe level for aquatic life. The toxic effect of mercury on aquatic organisms is not a good measure for use when taking into account possible effects on consumers of fish.

Until there is direct experimental evidence using species of wildlife such as ungulates and carnivores, the level for drinking water for livestock discussed in the following section, i.e. 0.003 mg/L, is recommended.

Agricultural Water Supply for Livestock

Introduction

In determining the guidelines for mercury concentration in the water supply for farm animals, two significant effects should be considered: (i) the effect of the mercury on the animal itself and (ii) a secondary effect, the mercury accumulation in the animal body, parts of which will be used for human consumption.

In studying the mercury concentration in the water supply, the ingestion of mercury from other sources, mainly air and feed, must also be considered.

Toxicity of Mercury to Farm Animals

The toxicity of mercury depends on its chemical form. All forms of mercury, including elemental mercury, its inorganic and organic salts, and the organic compounds, are toxic (NAS/NAE 1973, Hugunin and Bradley 1975a). The most toxic are the alkyl compounds (Neathery and Miller 1975; Schroeder and Mitchener 1975), especially methylmercury. This is probably due to their ability to cross cell membranes, which results in higher absorbability. The absorption of methylmercury chloride via the gastrointestinal tract of a lactating cow and goat, for example, was around 70% of the dose administered, while for mercuric chloride the corresponding value was 14% (Sell and Davison 1975). The slower elimination rate of the absorbed methylmercury compared with the inorganic mercury also contributed to the higher toxicity of the former. It has been estimated that the half-life of methylmercury is 70 days in humans and 88 days in rats compared with 30 and 13 days, respectively, for mercuric chloride (Neathery and Miller 1975).

The symptoms of mercury poisoning depend on the route of entry, chemical form, time of exposure, and the amount ingested. Observed symptoms were anorexia, progressive incoordination, excessive salivation, progressive weakness followed by prostration, complete incoordination, and finally death (Neathery and Miller 1975; Palmer *et al.* 1973). In experimental studies, the main symptom followed is the loss of weight (Schroeder and Mitchener 1975; March *et al.* 1974).

Tissue Distribution and Critical Levels

The tissue distribution depends on the chemical form, the entry route, the dosage, and the animal species (Hugunin and Bradley 1975a; Neathery and Miller 1975;

Nord. Hyg. Tidskr. 1971). Mercury, especially alkyl mercury, is found in all tissues. The critical tissue seems to be the nervous system (Hugunin and Bradley 1975a; *Nord. Hyg. Tidskr.* 1971, p. 155). In cases of intoxication with neurological symptoms or death, mercury concentrations in the brains of mammals ranged from 0.01 mg/kg (rat) to 61 mg/kg (cat) (*Nord. Hyg. Tidskr.* 1971, pp. 89, 164). More specifically, the brain concentrations of alkyl mercury in some farm animals with signs of poisoning were approximately 2 mg/kg in turkey, 8 mg/kg in cattle, and 10 mg/kg in sheep (Palmer *et al.* 1973). The concentration in the brain tissue of humans exposed to mercury while working ranged from 0.1 to 1.5 mg/kg, while the concentration in the brain tissue of populations living in an exposed area ranged from 0.02 to 0.08 mg/kg (Kosta *et al.* 1974).

Although the critical level depends to a large extent on the animal species and although different experimental conditions make it very difficult to reach any exact conclusions for domestic animals, the "working" number for the critical level of mercury in the brain tissue is 1.0 mg/kg.

Besides the critical level in the nervous system, a secondary effect, i.e., the accumulation of mercury in other tissues that will be used for human consumption, must also be considered. Present food guidelines allow for a maximum of 0.5 mg/kg or less of mercury in food for human consumption (see for example Hugunin and Bradley 1975b; NHW 1971).

In farm animals, the organ that showed the highest mercury concentration was the kidney (Neathery and Miller 1975; Palmer *et al.* 1973; *Nord. Hyg. Tidskr.* 1971) with the possible exception of chickens in which the liver and the kidney were found to have similar concentrations (March *et al.* 1974; Al-Fayadh *et al.* 1976).

Kidney/brain mercury concentration ratios ranging from 8 to 17 have been found in cattle (Neathery and Miller 1975; Palmer *et al.* 1973), 13 to 23 in sheep, 4 to 5 in turkey (Palmer *et al.* 1973), 2 to 4 in pig (*Nord. Hyg. Tidskr.* 1971, p. 89), and 7 in calves (Neathery and Miller 1975). In chickens, the liver/brain ratio ranged from 4 to 17 depending on the sex of the bird and the dosage of mercury (Al-Fayadh *et al.* 1976).

Antagonistic and Synergistic Effects

Dietary selenium (0.03 to 0.05 mg/kg Se) decreased the mercury concentration in various tissues of swine, and mercury toxicity signs did not appear in pigs given mercury and additional dietary selenium (Fros[illegible] *et al.* 1974). The same protective effect of selenium towards methylmercury intoxication was found in rats (Ohi *et al.* 1976; Hill 1975) and chicks (Hill 1973).

Experiments with sliced newborn rat cerebella in culture showed that the protective effect of sodium selenate and selenite against the neurotoxicity of methyl-mercury was additive with that of vitamin E (Kasuya 1976). In experiments with guinea pigs, toxic effects of mercury were not affected by large doses of vitamin C (ascorbic acid). Administration of large doses of vitamin C resulted in an increased deposition of mercury in the liver and kidney, while mercury reduced the concentration of ascorbic acid in the brain, adrenals, and spleen of animals receiving maintenance doses of the vitamin (Blackstone *et al.* 1974).

It was also found that chronic mercury intoxication and environmental conditions (temperature and air velocity) had a synergistic effect on chickens. The thermoregulation mechanism was impaired by nontoxic levels of mercury, and the mean survival time of chickens was the lowest for mercury-treated birds subjected to high air flow (Thaxton *et al.* 1975).

Criteria for Mercury Concentration in Water

The examination of the kidney/brain mercury concentration ratios, together with the 0.5 mg/kg maximum permissible concentration in food for human consumption, led to the conclusion that the limiting factor for a criterion value for mercury in the water supply for farm animals should be based on the mercury accumulation in kidney (or liver for chickens) rather than on its accumulation in the nervous system.

The literature data show that a direct relationship exists between the amount of mercury ingested and its concentration in various organs (Palmer *et al.* 1973; *Nord. Hyg. Tidskr.* 1971; Al-Fayadh *et al.* 1976; Kitamura *et al.* 1976). The scarcity of the data and the variation in experimental conditions preclude at this time any exact conclusions. It has been shown, however, that minute amounts of mercury ingested on a regular basis lead to relatively high mercury concentrations in the kidney or liver. In cattle, the ingestion of 0.48 mg per day per kilogram of body weight of mercury (as ethylmercury p-toluene sulphonanilide) resulted in kidney concentrations of approximately 100 mg/kg after 27 days. In sheep, an equal dose led to kidney concentrations of 120 to 210 mg/kg. The background "natural" concentrations of mercury in the kidney in the experiments with cattle, sheep or turkeys were 0.5 mg/kg or greater (Palmer *et al.* 1973).

In chickens, administration of approximately 1 μg or less of mercury (as fish meal mixed with the regular feed) per day per kilogram of body weight for a long period of time (one year) resulted in mercury concentrations in the kidney of 0.2 to 0.4 mg/kg (March *et al.* 1974).

Values quoted in NAS/NAE (1973) indicated that an ingestion of mercury of 2.3 μg per day kilogram of body weight led to blood concentrations of below 0.1 mg/L.

If one refers to the various kidney/brain mercury ratios and if a maximum mercury concentration of 0.5 mg/kg in kidney, liver or other edible animal tissue is permitted, then the maximum concentration of mercury in blood should be only approximately 0.005 mg/L (assuming a blood/brain ratio of 0.2 and a brain/tissue ratio of 0.05). This concentration is one twentieth of that recommended by the U.S. Environmental Protection Agency (NAS/NAE 1973). No data were found in the literature which permitted an estimation of the mercury intake which would lead to these maximum permissible concentrations. The situation is further complicated by the fact that in establishing a criterion for mercury in water supplies, the other sources of mercury, e.g., air and feed, must also be considered.

The data quoted above for chickens show that to avoid tissue concentrations of mercury greater than 0.5 mg/kg the maximum daily mercury intake should be approximately 0.001 mg/kg of body weight. Assuming that 25% of the mercury intake comes from the water supply and that the water intake of a bird is approximately 8% of its body weight daily (NAS/NAE 1973, pp. 304, 314), the concentration of mercury as methylmercury in water should be no more than 0.003 mg/L. The same value is obtained if an approximate method of calculation is used. For detailed calculations see Appendix A.

Recommended Objective for Livestock

The concentration of total mercury in the water supply for livestock should not exceed 0.003 mg/L. It is estimated that if this value is not exceeded, the normal water consumption should not contribute more than 0.2 mg/kg to the accumulation of mercury in the edible tissues of domestic animals or birds.

Water for Irrigation

As in the case of water supply for livestock, two significant effects have been considered in establishing a guideline for the mercury concentration in irrigation water: (i) a primary effect of toxicity to the plant itself, and (ii) a secondary effect of accumulation in parts of the plant that will be used for human or animal consumption.

Sources of Mercury to Plants

There are three pathways by which mercury can enter the plant tissue.

(i) Mercury present in soil from atmospheric deposition, from pesticidal compounds applied to the soil or of natural origin can enter via the plant roots.

(ii) Mercury from foliar sprays, dust or rain can penetrate the leaf tissue.

(iii) Mercury from irrigation water can enter the root system.

Mercury is present in the soil, atmosphere and water in its elemental metallic form or as inorganic or organic compounds.

The mercury concentration of a variety of soil samples collected in Canada ranged between <0.005 and 1.7 mg/kg with an average of 0.086 mg/kg (dry weight). One value of 14 mg/kg in a mercuriferous belt on an island in the Mackenzie River was also reported (McKeague and Kloosterman 1974). In southwestern Ontario mercury concentration in soils ranged from 0.003 to 0.100 mg/kg (Whitby *et al.* 1978). Frank *et al.* (1976) reported a range of 0.01 to 0.78 mg/kg and a mean of 0.18 mg/kg for mercury concentration in agricultural soils from Ontario. The mercury content of apple orchard soils, where phenylmercuric acetate was used over a 20-year period, was slightly higher, ranging from 0.03 to 1.14 mg/kg with an average of 0.33 mg/kg. Mills and Zwarich (1975) reported a range of 0.015 to 0.076 mg/kg and an average of 0.037 mg/kg for agricultural soils in southern and western Manitoba. The concentration of total mercury in Canadian prairie soils ranges from 0.01 to 0.04 mg/kg (Stewart *et al.* 1975). Note that the concentration of mercury in surface waters of Western Canada ranges from 0.05 to less than 1 μg/L, with most of the results at the lower end of this range (Environment Canada).

There was no detectable accumulation of mercury in urban areas of Winnipeg as compared to rural soils, near highways, or in farm soils from use of methyl mercurials over a 40-year period for treatment of seed grain (Mills and Zwarich 1975; Frank *et al.* 1976). Very high mercury concentrations with average of 53 mg/kg (range 27 to 65 mg/kg) were reported in the top 5 cm of soil collected from the greens of some golf courses in the Ottawa region. Mercurial compounds were widely used to control diseases of turf grass for a period of approximately 50 years, but this use was discontinued about 8 years before the soil samples were collected. The average mercury concentration in the turf grass itself was 57 mg/kg (range 24 to 120 mg/kg).

Several studies reported good direct correlations between the mercury concentration in soil and its organic matter content and an inverse correlation with depth (MacLean *et al.* 1973; Whitby *et al.* 1978; McKeague and Kloosterman 1974). Similarly it has been reported that in an incubation experiment in which 5 mg of phenylmercuric acetate was added per kilogram of soil, sandy soils lost 29 to 59% of the mercury by volatilization during a 2-month

period, while only 3% was lost from peat and 8% from a loam soil (MacLean 1974).

Methylation of the inorganic forms occurs both in water and soil, although only a very small part, 1×10^{-5}, of the mercuric ion applied to a soil was found in the form of methylmercury (Rogers 1976). It is not clear whether or not this is due to an unfavourable equilibrium towards the formation of methylmercury, to biotic demethylation, or to the fact that mercury in soil is bound in such a way that most of it is not available for methylation.

Toxicity to Crops

Plants seem to take up very little of the mercury present in soil. Experimental studies with plants grown on soil containing 10 mg/kg mercury showed that the resulting concentrations were 0.01 to 0.04 mg/kg in grain and seed, and 0.2 to 0.4 mg/kg in straw (wheat, rape). The foliage of alfalfa had a mercury concentration of 0.2 mg/kg, while the concentrations in the main roots and the fine roots were 3.5 to 4.5 mg/kg, respectively (Stewart *et al.* 1975). Plants growing near mercury deposits were found to contain 0.5 to 3.5 mg Hg/kg (Lagerwerff 1972).

In most plants mercury concentrations range from 0.010 to 0.200 mg/kg. Lettuce grown in soils containing 0.090 to 27.5 mg Hg/kg had a mercury content ranging from 0.029 to 0.317 mg/kg (oven-dry basis). Similarly, lettuce grown in soils containing no detectable mercury and 6 mg Hg/kg had mercury concentrations of 0.023 and 0.033 mg/kg, respectively. A series of plants grown in a loam soil containing 5.24 mg Hg/kg had a mercury content ranging from non-detectable in tobacco stems to 0.444 mg/kg (oven-dry basis) in soybean vines. The control plants grown in the same soil but containing only 0.080 mg Hg/kg had mercury concentrations ranging from "non-detectable" (tobacco stems and corn) to 0.364 mg/kg (potato tops) (MacLean 1974).

The low tendency of plant tissue to accumulate mercury is also demonstrated by comparing the ratios of mercury content in the mercury-treated soil with ratios in control soil and that of resulting concentrations in the tissues of the plants grown in those soils. In the experiments quoted above (MacLean 1974) the increase in the mercury concentration in the test soils over the controls was greater than 6400% (or 5.16 mg Hg/kg), while the change in the mercury concentration in the plants ranged from a loss of 6% to an increase of only 109% over the respective controls (in absolute values the change ranged from a loss of 0.009 mg/kg in alfalfa to an increase of 0.194 mg/kg in soybean vines). Similarly in another case, the mercury content of treated soil was approximately 250 times the level of mercury in the control soil, but the mercury content of the treated plants was only four to six times higher than the plants grown in control soil (Stewart *et al.* 1975).

Carrots and certain species of mushroom have the ability to concentrate mercury from soil. Mushrooms of the *Agaricus edulis* species grown on soil containing 0.3 mg/kg mercury had a mercury concentration in the carpophores of 4 to 10 mg/kg dry weight, which represents a concentration factor of 13 to 33 (Stijve and Besson 1976).

No indications of damage to the crops due to mercury were found in any of the few studies available in this field.

Synergistic Effects

Lower pH values of soil led to higher mercury concentrations in plants (Lagerwerff 1972). This is in agreement with the general tendency of lower availability of toxic metals at pH values above 6.5. In order to be absorbed by the root system the metal should be in a soluble form; hence the insoluble hydroxides and carbonates that are formed at pH values greater than 6.5 decrease the soluble part of the toxic metal in soil (Spotswood and Raymer 1973).

Recommended Objective for Irrigation Water

No data were found in the literature which relate the concentration of mercury in water to that in crops. Plants, with few exceptions, do not concentrate mercury in their tissues. It has been estimated that most of the mercury available to plants is of soil origin, with only a small part coming from irrigation water.

No guideline is being proposed for mercury in irrigation water because of these considerations.

Recreation and Aesthetics

Recreational waters are used to varying degrees; some have large concentrations of summer or winterized cottages which may utilize the lake or river as a source of drinking water. The level of mercury in the natural water in these situations should not exceed 0.001 mg/L.

Fish and shellfish caught during sport fishing should not be consumed from waters that are known to be contaminated by mercury, as there is a very large biomagnification of mercury contamination in aquatic organisms. The level of mercury in fish which is assumed to be safe for human consumption is 0.5 mg/kg (wet weight basis). (See section on aquatic life).

Recommended Objective for Recreational Waters

The concentration of mercury in water should not exceed 0.001 mg/L total mercury in areas where cottage

water supplies are taken from a lake or river. (See section on aquatic life concerning mercury content of fish).

Industrial Water Supplies

Water quality requirements for industry vary widely, but most industries are capable of treating raw water to attain a quality that is acceptable to the industry in question. With respect to the level of mercury, the industries concerned would be those involved in food and drink processing.

Recommended Objective for Industry

Water that is incorporated in the final product(s) of the food and drink industries must meet the same mercury requirement as potable water, i.e., a maximum concentration of 0.001 mg/L.

In the canning industry, the cooling water should also meet the same requirement.

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APPENDIX A

CALCULATION OF ACCUMULATED MERCURY IN THE ANIMAL BODY

Accumulated mercury in the whole body can be calculated from Kitamura *et al.* (1976):

Accumulated mercury = Daily dose X biological half-life X 1.44

1. If the daily water intake of animals (birds) is 8% of their body weight (or 0.08 L/kg of body weight) and the mercury concentration in water is 3 μg/L, then the daily dose from water intake is 0.24 μg/kg of body weight.
2. Assuming a 70-day biological half-life:

   Accumulated mercury = 0.24 X 70 X 1.44 ≃ 25 μg/kg body weight
3. Assuming a mercury concentration ratio of 4 for kidney/whole body (March *et al.* 1974), the mercury concentration in the kidney is:

   25 μg/kg X 4 = 0.1 mg/kg
4. If water accounts for 25% of the total daily intake of mercury, the long-term kidney mercury concentration will be 0.4 mg/kg.